CLAIMS

1. A method for detecting superantigen activity in a biological sample, characterized in that a majority expansion of lymphocytes bearing a Vβ16 and/or Vβ17 determinant or a majority loss of lymphocytes bearing a Vβ16 and/or Vβ17 determinant is demonstrated.

2. The detection method as claimed in claim 1, characterized in that a majority expansion of lymphocytes bearing a Vβ16 determinant is demonstrated.

3. The detection method as claimed in claim 1, characterized in that a majority loss of lymphocytes bearing a Vβ16 determinant is demonstrated.

4. The method as claimed in claim 2, characterized in that a majority expansion of lymphocytes bearing a Vβ16 determinant and a co-expansion of lymphocytes bearing Vβs chosen from at least any one of Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, and preferably of Vβ3 and Vβ12, are demonstrated.

5. The method as claimed in claim 3, characterized in that a majority loss of lymphocytes bearing a Vβ16 determinant and of [sic] a co-decrease of lymphocytes bearing Vβs chosen from at least any one of Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, preferably at least any one of Vβ7, Vβ14 and Vβ17, and advantageously of Vβ7 and Vβ17, are demonstrated.

6. The method as claimed in any one of the preceding claims, characterized in that the biological sample originates from a patient suffering from an autoimmune disease, in particular multiple sclerosis.

7. The method for detecting superantigen activity as claimed in any one of claims 1 to 6, characterized in that:

(i) a culture supernatant of blood mononucleated cells or of choroid plexus cells or of leptomeningeal cells, said cells originating from patients suffering from an autoimmune disease or

suspected of having a risk of developing the disease, in particular multiple sclerosis, or of an established cell line, such as the cells of the PLI-2 cell line deposited at the ECACC on July 22, 1992, under the number 92072201 and the LM7PC cell line deposited at the ECACC on January 8, 1993, under the number 93010817, in accordance with the provisions of the Treaty of Budapest, is sampled, and

(ii) said culture supernatant, or a part of the culture supernatant is brought into contact with a series of cultures, preferably at least three, of blood mononucleated cells originating from healthy donors, and

(iii) said expansion and, optionally, a co-expansion, or said loss and, optionally, co-decrease of the blood mononucleated cells of step (ii) are detected.

8. The method as claimed in claim 7, characterized in that the blood mononucleated cells originating from patients suffering from multiple sclerosis (MS) are chosen from monocytes and B lymphocytes and the blood mononucleated cells originating from healthy donors are chosen from T lymphocytes.

9. The method for detecting superantigen activity as claimed in any one of claims 1 to 6, characterized in that:

(i) blood mononucleated cells are sampled, said cells originating from patients suffering from an autoimmune disease or from patients suspected of having a risk of developing an autoimmune disease, in particular MS, and from healthy individuals,

(ii) said blood mononucleated cells originating from patients or from healthy individuals are brought into contact with culture supernatants, or a fraction of culture supernatant, of cells chosen from blood mononucleated cells, choroid plexus cells and lepto-meningeal cells, and cells derived from established

cell lines, such as the cells of the PLI-2 cell line deposited at the ECACC on July 22, 1992, under the number 92072201 and the LM7PC cell line deposited at the ECACC on January 8, 1993, under the number 93010817, in accordance with the provisions of the Treaty of Budapest, and

(iii) said expansion and, optionally, co-expansion, or said loss and, optionally, co-decrease, using the blood mononucleated cells of step (i) are detected.

10. The method as claimed in claims 7, 8 and 9, characterized in that said expansion and, optionally, co-expansion is demonstrated using ligands, each ligand being specific for a determinant chosen from Vβ16, Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, preferably Vβ16, Vβ3 and Vβ12, and in that said loss and, optionally co-decrease is demonstrated using ligands, each ligand being specific for a determinant chosen from Vβ16, Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, preferably Vβ16, Vβ7, Vβ14 and Vβ17.

11. The method as claimed in claim 10, characterized in that the ligand is an antibody, preferably a monoclonal antibody or an antibody fragment.

12. The method as claimed in claim 7, 8 and 9, characterized in that in order to demonstrate said expansion and, optionally, co-expansion or said loss and, optionally, co-decrease, the following is carried out

(i) extraction of the total RNAs from the blood mononucleated cells which have been placed together with MS culture supernatant or a fraction of MS culture supernatant and together with control culture supernatant or a fraction of control culture supernatant,

(ii) reverse transcription of said RNAs,

(iii) amplification specific for each Vβ family using a given pair of primers,

(iv) labeling of the amplification products obtained, with any suitable label,

(v) electrophoresis of said amplification products and analysis of the electrophoretic profiles obtained, using a suitable detector.

13. The method as claimed in claim 12, characterized in that the blood mononucleated cells originating from patients suffering from MS are chosen from lymphocytes.

14. A method for detecting a pathological condition or a predisposition to a pathological condition, in a biological sample, characterized in that at least one of the following parameters is demonstrated:

superantigen activity, as defined in any one of claims 1 to 13,

stimulation of the production of cytokines, such as interleukin-6 (IL-6) and γ-interferon (γ-INF),

induction of cellular apoptosis.

15. The method as claimed in claim 14, characterized in that at least two of the parameters are detected in combination.

16. The method as claimed in claim 15, characterized in that superantigen activity and induction of apoptosis or superantigen activity and stimulation of the production of cytokines are detected.

17. The method as claimed in claim 14, characterized in that the three parameters are detected in combination.

18. The method as claimed in claim 7, 8 and 9, characterized in that the pathological condition is associated with an autoimmune disease, such as multiple sclerosis.

19. The method as claimed in any one of the preceding claims, characterized in that the

superantigen activity is induced directly or indirectly by an effector agent chosen from proteins and/or microorganisms and/or pathogenic agents.

20. The method as claimed in claim 19, characterized in that the microorganism is chosen from bacteria and retroviruses, preferably human retroviruses, and in particular the retrovirus is MSRV-1 (Multiple sclerosis retrovirus 1) and in particular the pathogenic agent is MSRV-2 (Multiple sclerosis retrovirus 2).

21. The method as claimed in claims 19 and 20, characterized in that the superantigen activity is induced by the envelope protein of MSRV-1 referenced in SEQ ID No. 2 or by a fragment of said protein.

22. The method as claimed in claims 19 and 20, characterized in that the superantigen activity is induced by the *env* gene of MSRV-1 referenced in SEQ ID No. 1 or a fragment of said gene.

23. A human retrovirus, in particular an endogenous retrovirus, which has superantigen activity and is associated with an autoimmune disease, characterized in that the retrovirus is MSRV-1 and in that the superantigen activity is induced by the expression of the *env* gene of MSRV-1 or of a fragment of said gene, in particular a fragment of said gene encoding at least one reading frame of the env protein of MSRV-1 (SEQ ID No. 2).

24. A human retrovirus, in particular an endogenous retrovirus, which has superantigen activity and is associated with an autoimmune disease, characterized in that the retrovirus is MSRV-1 and in that the superantigen activity is induced by the env protein of MSRV-1 or by a fragment of said protein, in particular by a fragment corresponding to at least one reading frame of said protein (SEQ ID No. 2).

25. A nucleic acid molecule comprising at least one or more fragment(s) of the RNA or of the DNA of the *env*

gene of MSRV-1, identified by SEQ ID No. 1, said fragment being at least 18 nucleotides, and preferably at least 24 nucleotides, in length.

26. The nucleic acid molecule as claimed in claim 25, comprising at least one fragment encoding at least one reading frame and optionally containing a stop codon.

27. The nucleic acid molecule as claimed in claim 26, encoding superantigen activity.

28. A polypeptide molecule, in particular protein or protein fragment comprising at least one or more fragment(s) of said env protein of MSRV-1 identified by SEQ ID No. 2, said fragment being at least 6 amino acids, and preferably at least 8 amino acids, in length.

29. The polypeptide molecule as claimed in claim 28, comprising at least one reading frame.

30. The polypeptide molecule as claimed in claim 29, having superantigen activity.

31. A vector comprising nucleic acid molecules as defined in claims 25 to 27.

32. A method for detecting superantigen activity in a biological sample from patients suffering from multiple sclerosis, characterized in that a majority expansion of lymphocytes bearing a Vβ7 determinant or a majority loss of lymphocytes bearing a Vβ7 determinant is demonstrated.

33. The method as claimed in claim 32, characterized in that:

(i) a culture supernatant of blood mononucleated cells or of choroid plexus cells or of leptomeningeal cells, said cells originating from patients suffering from an autoimmune disease or suspected of having a risk of developing the disease, in particular multiple sclerosis, or of an established cell line, such as the cells of the PLI-2 cell line deposited at the ECACC on July 22, 1992, under the

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number 92072201 and the LM7PC cell line deposited at the ECACC on January 8, 1993, under the number 93010817, in accordance with the provisions of the Treaty of Budapest, is sampled, and

(ii) said culture supernatant, or a part of the culture supernatant is brought into contact with a series of cultures, preferably at least three, of blood mononucleated cells originating from healthy donors, and

(iii) said expansion and, optionally, a co-expansion, or said loss and, optionally, co-decrease of the blood mononucleated cells of step (ii) are detected.

34. The method as claimed in claim 33, characterized in that the blood mononucleated cells originating from patients suffering from MS are chosen from B lymphocytes and monocytes and the blood mononucleated cells originating from healthy donors are chosen from T lymphocytes.

35. The method as claimed in claim 32, characterized in that:

(i) blood mononucleated cells are sampled, said cells originating from patients suffering from an autoimmune disease or from patients suspected of having a risk of developing an autoimmune disease, in particular MS, and from healthy individuals,

(ii) said blood mononucleated cells originating from patients or from healthy individuals are brought into contact with culture supernatants, or a fraction of culture supernatant, of cells chosen from blood mononucleated cells, choroid plexus cells and leptomeningeal cells, and cells derived from established cell lines, such as the cells of the PLI-2 cell line deposited at the ECACC on July 22, 1992, under the number 92072201 and the LM7PC cell line deposited at the ECACC on January 8, 1993, under the

number 93010817, in accordance with the provisions of the Treaty of Budapest, and

(iii) said expansion and, optionally, co-expansion, or said loss and, optionally, co-decrease, using the blood mononucleated cells of step (i) are detected.

36. The method as claimed in claim 32, characterized in that the superantigen activity is demonstrated according to a protocol as described in claims 10 to 12, using a ligand or amplification combined with electrophoresis.

37. The method for detecting superantigen activity as claimed in any one of claims 1 to 6, characterized in that

(i) a polypeptide, in particular a recombinant protein, as identified by SEQ ID No. 2, or a fragment of said polypeptide or of said protein, is produced or synthesized,

(ii) said polypeptide or said protein is brought into contact with a series of cultures, preferably at least three, of blood mononucleated cells originating from healthy donors, and

(iii) said expansion and, optionally, a co-expansion, or said loss and, optionally, co-decrease, of the blood mononucleated cells of step (ii) are detected.

38. The method for detecting superantigen activity as claimed in any one of claims 1 to 6, characterized in that:

(i) blood mononucleated cells are sampled, said cells originating from patients suffering from an autoimmune disease or from patients suspected of having a risk of developing an autoimmune disease, in particular MS, and from healthy individuals,

(ii) said blood mononucleated cells originating from patients or from healthy individuals are brought into contact with a polypeptide or a recombinant

protein, as identified in SEQ ID No. 2, or a fragment of said polypeptide or of said protein, and

(iii) said expansion and, optionally, co-expansion, or said loss and, optionally, co-decrease, using the blood mononucleated cells of step (i) are detected.

39. The method as claimed in claim 38, characterized in that a polypeptide as defined in claims 28 to 30 is used.

40. The method as claimed in claim 37 or 38, characterized in that said polypeptide is encoded by a nucleic acid as defined in claims 26 to 27 or a vector as claimed in claim 31.

41. A method for evaluating the effectiveness of an agent or of a composition in inhibiting superantigen activity in a biological sample, characterized in that

i) a culture supernatant of blood mononucleated cells, or of choroid plexus cells or of leptomeningeal cells, said cells originating from patients suffering from an autoimmune disease, in particular MS, or of cells of an established cell line, such as the cells of the PLI-2 line and the LM7PC line, is sampled,

ii) said supernatant, or a part of the culture supernatant, is brought into contact with a series of cultures, preferably at least three, of blood mononucleated cells originating from healthy donors, in the presence of said agent or of said composition at predetermined doses, and

(iii) the inhibition of said expansion and, optionally, co-expansion, or the inhibition of said loss and, optionally, co-decrease, of the lymphocytes bearing at least one determinant chosen from Vβ16, Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, in particular Vβ16 and/or Vβ17, Vβ16, Vβ3 and Vβ12 or Vβ16, Vβ7, Vβ14 and Vβ17, particularly Vβ16, Vβ7 and Vβ17, are detected using a ligand as described in claims 10 and 11 or

amplification combined with electrophoresis as described in claim 12.

42. A method for evaluating the effectiveness of an agent or of a composition in inhibiting superantigen activity in a biological sample, characterized in that

(i) a polypeptide, in particular a recombinant protein is produced or synthesized,

(ii) said polypeptide or recombinant protein is brought into contact with a series of cultures, preferably at least three, of blood mononucleated cells originating from healthy donors, in the presence of said agent or of said composition at predetermined doses, and

(iii) the inhibition of said expansion and, optionally, co-expansion, or the inhibition of said loss and, optionally, co-decrease, of the lymphocytes bearing at least one determinant chosen from Vβ16, Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, in particular Vβ16 and/or Vβ17, Vβ16, Vβ3 and Vβ12 or Vβ16, Vβ7, Vβ14 and Vβ17, particularly Vβ16, Vβ7 and Vβ17, are detected using a ligand as described in claims 10 and 11 or amplification combined with electrophoresis as described in claim 12.

43. A method for evaluating the effectiveness of an agent or of a composition in inhibiting superantigen activity in a biological sample, characterized in that

(i) blood mononucleated cells are sampled, said cells originating from patients suffering from an autoimmune disease or suspected of having a risk of developing the disease, in particular MS, and from healthy individuals,

(ii) said blood mononucleated cells originating from patients or from healthy individuals are brought into contact with culture supernatants, or a fraction of culture supernatant, of cells chosen from blood mononucleated cells, choroid plexus cells, leptomeningeal cells and cells derived from established

cell lines, such as the cells of the PLI-2 cell line and the LM7PC cell line, and

(iii) the inhibition of said expansion and, optionally, co-expansion, or the inhibition of said loss and, optionally, co-decrease, of the lymphocytes bearing at least one determinant chosen from Vβ16, Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, in particular Vβ16 and/or Vβ17, Vβ16, Vβ3 and Vβ12 or Vβ16, Vβ7, Vβ14 and Vβ17, particularly Vβ16, Vβ7 and Vβ17, using the blood mononucleated cells of step (i), in the presence of said agent or of said composition at given doses, are detected using a ligand as described in claims 10 and 11 or amplification combined with electrophoresis as described in claim 12.

44. A method for evaluating the effectiveness of an agent or of a composition in inhibiting superantigen activity in a biological sample, characterized in that

(i) blood mononucleated cells are sampled, said cells originating from patients suffering from an autoimmune disease or suspected of having a risk of developing the disease, in particular MS, and from healthy individuals,

(ii) said blood mononucleated cells originating from patients or from healthy individuals are brought into contact with a polypeptide or a recombinant protein, and

(iii) the inhibition of said expansion and, optionally, co-expansion, or the inhibition of said loss and, optionally, co-decrease, of the lymphocytes bearing at least one determinant chosen from Vβ16, Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, in particular Vβ16 and/or Vβ17, Vβ16, Vβ3 and Vβ12 or Vβ16, Vβ7, Vβ14 and Vβ17, particularly Vβ16, Vβ7 and Vβ17, using the blood mononucleated cells of step (i), in the presence of said agent or of said composition at given doses, are detected using a ligand as described in claims 10

and 11 or amplification combined with electrophoresis as described in claim 12.

45. The method as claimed in any one of claims 41 to 44, characterized in that the cells originate from a patient suffering from an autoimmune disease, in particular multiple sclerosis.

46. The method as claimed in any one of claims 41 to 45, characterized in that the blood mononucleated cells originating from patients suffering from MS are chosen from B lymphocytes and monocytes.

47. A method for evaluating the prophylactic and/or therapeutic effectiveness of an agent or of a composition with respect to a pathological condition and/or to a predisposition to a pathological condition, characterized in that inhibition of superantigen activity in a biological sample is demonstrated as described in claims 41 to 46.

48. The method as claimed in claim 47, characterized in that the inhibition of the majority loss of lymphocytes bearing a Vβ16 determinant and the co-decrease of lymphocytes bearing Vβ7 and Vβ17 are demonstrated.

49. The method as claimed in ~~claim~~ 46, characterized in that the inhibition of the majority expansion of lymphocytes bearing a Vβ16 determinant and the co-expansion of lymphocytes bearing Vβ3 and Vβ12 are demonstrated.

50. The method as claimed in either of ~~claims~~ 48 and 49, characterized in that the cells originate from a patient suffering from an autoimmune disease, such as multiple sclerosis.

51. The method as claimed in claims 48 to 50, characterized in that the blood mononucleated cells originating from patients suffering from MS are chosen from B lymphocytes and monocytes.

52. A method for evaluating the prophylactic and/or therapeutic effectiveness of an agent or of a

composition with respect to a pathological condition and/or to a predisposition to a pathological condition, characterized in that inhibition of superantigen activity in a biological sample is demonstrated as described in claims 48 to 51.

53. Composition for therapeutic and/or prophylactic use, characterized in that it comprises, inter alia, a therapeutic agent capable of inhibiting superantigen activity in a biological sample, as defined in claims 1 to 6, optionally in combination with a pharmaceutically acceptable excipient and/or adjuvant and/or diluent.

54. The composition as claimed in claim 53, characterized in that the therapeutic agent is an antiviral agent, more particularly an antiretroviral agent, in particular a human antiretroviral agent, preferably an anti-MSRV1 agent, such as an inhibitor of the replication cycle and/or of the expression of a retrovirus, such as an anti-retroviral protein antibody, in particular an anti-envelope antibody, such as antisense oligonucleotides, more particularly which block retroviral expression.

55. Composition as claimed in claim 53, characterized in that the therapeutic agent is chosen from a natural molecule and/or a recombinant molecule, or a fragment of said molecules, the protein sequence of which corresponds to the sequence of the Vβ16 and/or Vβ17 molecules, preferably the Vβ16 molecule, optionally in combination with one or more natural and/or recombinant molecules, or a fragment of said molecules, the protein sequence of which corresponds to the sequence of the Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22 molecules, and preferentially of the Vβ3 and Vβ12 molecules.

56. Composition as claimed in claim 53, characterized in that the therapeutic agent is chosen from a natural molecule and/or a recombinant molecule, or a fragment of said molecules, the protein sequence

of which corresponds to the sequence of the Vβ16 and/or Vβ17 molecules, and optionally in combination with one or more natural and/or recombinant molecules or a fragment of said modules, the protein sequence of which corresponds to the Vβ7, Vβ14 and Vβ17 molecules, and preferentially of the Vβ7 and Vβ17 molecules.

57. The composition as claimed in claim 53, characterized in that the therapeutic agent is chosen from the natural and/or recombinant and/or synthetic molecules, or a fragment of said molecules, which encode the molecules as defined in claims 53 to 56.

58. The composition as claimed in claim 57, characterized in that the therapeutic and/or prophylactic agent is chosen from the therapeutic genes comprising DNA and/or RNA molecules.

59. The prophylactic and/or therapeutic composition as claimed in claim 53, characterized in that the prophylactic and/or therapeutic agent is chosen from antisense oligonucleotides and antigene oligonucleotides.

60. The prophylactic and/or therapeutic composition as claimed in claim 53, characterized in that the prophylactic and/or therapeutic agent is chosen from at least one ligand capable of interacting with Vβ16 and/or Vβ17, in particular Vβ16, optionally in combination with at least one ligand capable of interacting with at least one of Vβ2, Vβ3, Vβ7, Vβ8, Vβ12, Vβ14, Vβ17 and Vβ22, and preferentially Vβ3 and Vβ12.

61. The composition as claimed in claim 60, characterized in that the ligand is capable of interacting with a retrovirus, in particular a human retrovirus, such as MSRV-1, its proteins and/or its nucleic acids.

62. The composition as claimed in claim 60, characterized in that the ligand is an antiviral agent, more particularly an antiviral agent, in particular a

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human antiviral agent, preferably an anti-MSRV1 agent, such as an inhibitor of the replication cycle and/or of the expression of a retrovirus, such as an anti-retroviral protein antibody, in particular an anti-envelope antibody, such as antisense oligonucleotides, more particularly which block retroviral expression.

63. The composition as claimed in claim 60, characterized in that the ligand is chosen from antibodies, preferably monoclonal antibodies and anti-receptors for the T-cell receptors (TCRs) of the various Vβs.

64. The composition as claimed in claim 61, characterized in that the ligand is chosen from anti-MSRV-1 antibodies, preferably monoclonal antibodies.

65. The prophylactic and/or therapeutic composition as claimed in claim 53, characterized in that the prophylactic and/or therapeutic agent is chosen from at least one ligand capable of interacting with Vβ16 and/or Vβ17, optionally in combination with at least one ligand capable of interacting with at least one of Vβ7, Vβ14, vβ17 and Vβ22, and preferentially Vβ7 and Vβ17.

66. [sic] The composition as claimed in claim 60, characterized in that the ligand is capable of interacting with a retrovirus, in particular a human retrovirus, such as MSRV-1, its proteins and/or its nucleic acids.

67. The composition as claimed in claim 63 or 65, characterized in that the ligand is chosen from antibodies, preferably monoclonal antibodies and anti-receptors for the TCRs of the various Vβs.

68. The composition as claimed in claim 63 or 64, characterized in that the ligand is chosen from anti-MSRV-1 antibodies, preferably monoclonal antibodies.

69. The composition as claimed in claim 53, characterized in that the therapeutic and/or prophylactic agent is an agent capable of blocking the

interaction of the superantigen with the antigen-presenting cells.

70. A therapeutic and/or prophylactic composition, characterized in that the therapeutic and/or prophylactic agent is chosen from at least one cell, preferably a cell of mammalian origin, genetically modified *in vitro* with a therapeutic agent which consists of at least one nucleic acid molecule encoding at least one molecule, the protein sequence of which corresponds to the sequence encoding the molecules as defined in claims 1 to 5, 10, 20 to 31 and 53 to 59, in particular a DNA and/or RNA molecule.

71. The composition as claimed in claim 53, characterized in that the therapeutic and/or prophylactic agent is chosen from at least one cell, preferably a cell of mammalian origin, genetically modified *in vitro* with a therapeutic agent which consists of at least one nucleic acid molecule encoding at least one ligand as defined in claims 60 to 69, in particular a DNA and/or RNA molecule.

72. The use of a composition as defined in claims 53 to 71, for the prophylaxis and/or the treatment of a pathological condition, in particular an autoimmune disease such as multiple sclerosis.

73. A method for identifying substances capable of blocking the transcription and/or the translation of a human retrovirus, in particular a retrovirus which is endogenous, as defined in claims 23 and 24, and which has superantigen activity, said superantigen activity being associated with an autoimmune disease, according to which,

the substance is brought into contact with cells expressing a retroviral polypeptide as defined in claims 28 to 30 which has superantigen activity, and

a loss or decrease of the superantigen activity is detected as described in claims 1 to 6.

74. A kit for screening substances capable of blocking the superantigen activity of a retrovirus, in particular an endogenous human retrovirus, associated with an autoimmune disease, or capable of blocking the transcription and/or the translation of said retrovirus, comprising:

cells expressing, at their surface, class II MHC products, transformed with and functionally expressing a retroviral superantigen,

cells bearing receptor chains having one or more Vβs stimulated by the retroviral superantigen, and

means for detecting a loss or decrease of the superantigen activity as described in claims 1 to 6.

75. The use of substances capable of inhibiting a function of a human retrovirus, in particular an endogenous retrovirus, for preparing a medicinal product for use in therapy and/or prevention of an autoimmune disease associated with a retroviral superantigen, in particular associated with MS.

76. The use as claimed in claim 75, in which the substance is 3'-azido-3'-deoxythymidine (AZT) or dideoxyinosine (DDI).

77. The use of substances capable of inhibiting the superantigen function of a human retrovirus, in particular an endogenous retrovirus, for preparing a medicinal product for use in the therapy of an autoimmune disease associated with a retroviral superantigen, in particular associated with MS.